Amendments to the Claims:

This listing of claims will replace all prior versions and listings of claims in the application.

Listing of Claims:

1. (previously presented) An antibody selected from the group consisting of anti-EG-VEGF monoclonal antibodies 1C6.1H6.1D7, 2A3.1C5.1F3, 2A8.1H4.1E7 and 4H9.1A7.1H6.

2. (previously presented) An antibody that binds essentially the same epitope of EG-VEGF bound by an antibody selected from the group consisting of anti-EG-VEGF monoclonal antibodies 1C6.1H6.1D7, 2A3.1C5.1F3, 2A8.1H4.1E7 and 4H9.1A7.1H6.

3. (original) The antibody of claim 2 which is an antibody fragment.

4. (original) The antibody of claim 3 selected from the group consisting of Fab, Fab', $F(ab)_2$, and Fv fragments.

5. (original) The antibody of claim 2 which is a chimeric antibody.

6. (original) The antibody of claim 2 which is humanized.

7. (original) The antibody of claim 2 which is human.

8. (original) The antibody of claim 2 which is a bispecific antibody.

9. (original) The antibody of claim 8 wherein said bispecific antibody has binding specificity for VEGF.

10. (original) The antibody of claim 2 which is detectably labeled.

11. (original) A composition of matter comprising (a) an EG-VEGF polypeptide, (b) an agonist of an EG-VEGF polypeptide, or (c) an antagonist of an EG-VEGF polypeptide, in admixture with a pharmaceutically acceptable carrier.

12. (original) The composition of claim 11 wherein said EG-VEGF polypeptide is a native sequence EG-VEGF.

13. (original) The composition of claim 12 wherein said native sequence EG-VEGF is human.

14. (original) The composition of claim 11 wherein said agonist or antagonist is an anti-EG-VEGF-antibody.

15. (previously presented) The composition of claim 14 wherein said antagonist is an anti-EG-VEGF antibody selected from the group consisting of monoclonal antibodies 1C6.1H6.1D7, 2A3.1C5.1F3, 2A8.1H4.1E7 and 4H9.1A7.1H6.

16. (previously presented) The composition of claim 14 wherein said antagonist is an anti-EG-VEGF antibody that binds essentially the same epitope of EG-VEGF bound by an antibody selected from the group consisting of anti-EG-VEGF monoclonal antibodies 1C6.1H6.1D7, 2A3.1C5.1F3, 2A8.1H4.1E7 and 4H9.1A7.1H6.

17. (original) The composition of claim 11 wherein said agonist or antagonist is an anti-EG-VEGF antibody fragment.

18. (original) The composition of claim 17 wherein said antibody fragment is selected from the group consisting of Fab, FAb', $F(ab)_2$, and Fv fragments.

19. (original) The composition of claim 11 wherein said antagonist is an antisense molecule.

20. (original) The composition of claim 11 further comprising a vascular endothelial growth factor (VEGF), or an agonist or antagonist thereof.

21. (original) The composition of claim 20 wherein said VEGF is a native sequence VEGF polypeptide.

22. (original) The composition of claim 21 wherein said native sequence VEGF is human.

23. (previously presented) An article of manufacture, comprising:

a container;

a label on the container; and

a composition comprising an anti-EG-VEGF antibody binding essentially the same epitope as an antibody selected from the group consisting of monoclonal antibodies 1C6.1H6.1D7, 2A3.1C5.1F3, 2A8.1H4.1E7 and 4H9.1A7.1H6.

24. (previously presented) The article of manufacture of claim 23 comprising an anti-EG-VEGF antibody selected from the group consisting of monoclonal antibodies 1C6.1H6.1D7, 2A3.1C5.1F3, 2A8.1H4.1E7 and 4H9.1A7.1H6.

25. (original) A method for identifying a compound that modulates a biological activity of EG-VEGF, comprising the steps of:

a) contacting a candidate compound with EG-VEGF; and

b) determining an alteration in said biological activity of EG-VEGF.

26. (original) The method of claim 25 wherein said compound inhibits a biological activity of said EG-VEGF.

27. (original) The method of claim 25 wherein said compound enhances a biological activity of said EG-VEGF.

28. (original) The method of claim 25 wherein said biological activity is the ability to induce phosphorylation of a kinase involved in cell proliferation or survival.

29. (original) The method of claim 28 wherein said kinase is a MAP kinase.

30. (original) The method of claim 29 wherein said MAP kinase is ERK1 or ERK2

31. (original) The method of claim 25 wherein said biological activity is the ability to induce phosphorylation of Akt or eNOS.

32. (original) The method of claim 25 wherein said biological activity is the ability to stimulate cell proliferation.

33. (original) The method of claim 25 wherein said biological activity is the induction of chemotaxis.

34. (original) The method of claim 25 wherein said biological activity is the induction of angiogenesis.

35. (original) The method of claim 25 wherein said biological activity is the induction of cell differentiation.

36. (original) The method of claim 25 wherein said biological activity is the induction of endothelial cell fenestration.

37. (original) The method of claim 25 wherein said biological activity is the enhancement of endothelial cell survival.

38. (original) The method of claim 25 wherein said candidate compound is contacted with a whole cell or a cell membrane fraction expressing the coding sequence of EG-VEGF.

39. (original) The method of claim 38 wherein said cell is a recombinant host cell engineered to express said EG-VEGF.

40. (original) The method of claim 25 wherein said candidate compound is contacted with an isolated EG-VEGF.

41. (original) The method of claim 40 wherein said EG-VEGF is immobilized on a solid support.

42. (original) A compound identified by the method of claim 25.

43. (original) A method of inducing cell proliferation, comprising contacting said cells with EG-VEGF in an amount effective to induce proliferation of said cells.

44. (original) The method of claim 43 wherein said cells are endothelial cells.

45. (original) The method of claim 44 wherein said endothelial cells are steroidogenic endothelial cells.

46. (original) The method of claim 45 wherein said endothelial cells are cells of a steroidogenic gland.

47. (original) The method of claim 43 further comprising contacting said cells with VEGF.

48. (original) A method of inducing chemotaxis in cells, comprising contacting said cells with EG-VEGF in an amount effective to induce chemotaxis.

49. (original) The method of claim 48 wherein said cells are endothelial cells.

50. (original) The method of claim 49 wherein said cells are steroidogenic endothelial cells.

51. (original) The method of claim 50 wherein said cells are endothelial cells of a steroidogenic gland.

52. (original) The method of claim 48 further comprising contacting said cells with VEGF.

53. (original) A method of enhancing cell survival comprising contacting cells with EG-VEGF in an amount effective to enhance cell survival.

54. (original) The method of claim 53 wherein said cells are endothelial cells.

55. (original) The method of claim 54 wherein said cells are steroidogenic endothelial cells.

56. (original) The method of claim 55 wherein said cells are endothelial cells of a steroidogenic gland.

57. (original) The method of claim 53 further comprising contacting said cells with VEGF.

58. (original) A method of inhibiting endothelial cell proliferation, comprising contacting said cells with an EG-VEGF antagonist in an amount effective to inhibit cell proliferation.

59. (previously presented) The method of claim 58 wherein said EG-VEGF antagonist is an anti-EG-VEGF antibody selected from the group consisting of monoclonal antibodies 1C6.1H6.1D7, 2A3.1C5.1F3, 2A8.1H4.1E7 and 4H9.1A7.1H6.

60. (original) A method of inhibiting chemotaxis in endothelial cells, comprising contacting said cells with an EG-VEGF antagonist in an amount effective to inhibit chemotaxis.

61. (previously presented) The method of claim 60 wherein said EG-VEGF antagonist is an anti-EG-VEGF antibody selected from the group consisting of monoclonal antibodies 1C6.1H6.1D7, 2A3.1C5.1F3, 2A8.1H4.1E7 and 4H9.1A7.1H6.

62. (new) An antagonist of EG-VEGF polypeptide wherein the antagonist inhibits EG-VEGF polypeptide induced proliferation of adrenal cortex-derived endothelial cells.

63. (new) An antagonist of EG-VEGF polypeptide wherein the antagonist is an antibody, small molecule, peptide fragment, or antisense molecule.

64. (new) The antagonist of claim 62, wherein the antagonist is an antibody.

65. (new) The antagonist of claim 64, wherein the antibody is an antibody fragment.

66. (new) The antagonist of claim 65, wherein the antibody fragment is a Fab, Fab', $F(ab)_2$, or Fv fragment.

67. (new) The antibody of claim 64, wherein the antibody is
a) a chimeric antibody;
b) a single chain antibody;
c) a bispecific antibody; or
d) a humanized antibody.

68. (new) The antibody of claim 64, wherein the antibody is polyclonal.

69. (new) The antibody of claim 64, wherein the antibody is monoclonal.

70. (new) The antibody of claim 69, wherein the antibody is 1C6.1H6.1D7.

71. (new) The antibody of claim 69, wherein the antibody is 2A3.1C5.1F3.

72. (new) The antibody of claim 69, wherein the antibody is 2A8.1H4.1E7.

73. (new) The antibody of claim 69, wherein the antibody is 4H9.1A7.1H6.

74. (new) The antagonist of claim 62, wherein the EG-VEGF polypeptide is a native sequence EG-VEGF.

75. (new) The antagonist of claim 74, wherein the native sequence EG-VEGF is human.

76. (new) The antagonist of claim 62, wherein the EG-VEGF polypeptide comprises an amino acid sequence having at least about 80% identity to SEQ ID NO:2, wherein the polypeptide promotes proliferation of adrenal cortex-derived capillary endothelial cells.

77. (new) The antagonist of claim 76, wherein the amino acid sequence comprises amino acid residues 1 to 105 of SEQ ID NO:2.

78. (new) The antagonist of claim 76, wherein the amino acid sequence comprises amino acid residues 20 to 105 of SEQ ID NO:2.

79. (new) The antagonist of claim 76, wherein the amino acid sequence comprises amino acid residues X to 105 of SEQ ID NO:2, wherein X is an amino acid residue from 14 to 24 of SEQ ID NO:2